Pneumonia in HIV-Infected Patients

Seda Tural Önür[1], Levent Dalar[2], Sinem İliaz[3], Arzu Didem Yalçın[4,5]

[1]Clinic of Chest Diseases, Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital, İstanbul, Turkey
[2]Department of Chest Diseases, İstanbul Bilim University School of Medicine, İstanbul, Turkey
[3]Department of Chest Diseases, Koç University, İstanbul, Turkey
[4]Academia Sinica, Genomics Research Center, Internal Medicine, Allergy and Clinical Immunology, Taipei, Taiwan
[5]Clinic of Allergy and Clinical Immunology, Antalya Training and Research Hospital, Antalya, Turkey

Abstract

Acquired immune deficiency syndrome (AIDS) is an immune system disease caused by the human immunodeficiency virus (HIV). The purpose of this review is to investigate the correlation between an immune system destroyed by HIV and the frequency of pneumonia. Observational studies show that respiratory diseases are among the most common infections observed in HIV-infected patients. In addition, pneumonia is the leading cause of morbidity and mortality in HIV-infected patients. According to articles in literature, in addition to antiretroviral therapy (ART) or highly active antiretroviral therapy (HAART), the use of prophylaxis provides favorable results for the treatment of pneumonia. Here we conduct a systematic literature review to determine the pathogenesis and causative agents of bacterial pneumonia, tuberculosis (TB), nontuberculous mycobacterial disease, fungal pneumonia, *Pneumocystis* pneumonia, viral pneumonia and parasitic infections and the prophylaxis in addition to ART and HAART for treatment. *Pneumococcus*-based polysaccharide vaccine is recommended to avoid some type of specific bacterial pneumonia.

Keywords: HIV, infection, pneumonia

INTRODUCTION

Human immunodeficiency virus (HIV) targets the CD4 T-lymphocyte or T cells. As stated previously, pulmonary diseases are the main causes of morbidity and mortality among HIV-infected patients (1). People can be infected by HIV even years before they get acquired immune deficiency syndrome (AIDS) (2). The field of lung diseases in HIV-infected patients comprises both HIV-related and non-HIV-related situations. The HIV-associated lung situations consist of opportunistic infections (OIS) and malignancies. OIs are caused by bacterial, mycobacterial, fungal, viral, and parasitic agents (1, 3).

Respiratory symptoms include cough, dyspnea, and pleuritic chest pain either alone or in combination. Cough may or may not secrete clear phlegm/sputum, purulent sputum, blood-streaked sputum, or even light hemoptysis. Dyspnea may be mild or severe and appear when the body is at rest. Constitutional complaints may also show up. In addition, extrapulmonary symptoms (e.g., headache) may be observed and could aid in differentiating the various OIs and neoplasms (3).

This author's first-hand experience indicates that these types of pneumonia are often differentiated from each other according to the absence of purulent sputum and the duration of respiratory symptoms (4).

The presence of systemic hypotension would be concerning for a fulminant disease process. Predictors of mortality are age, recent drug injection, total bilirubin, serum albumin lower than 3 g/dL, and alveolar–arterial oxygen gradient greater than or equal to 50 mmHg for *Pneumocystis carinii* pneumonia (PCP) (5).

Our review elucidates the pathogenesis and causative agents of bacterial pneumonia, tuberculosis (TB), nontuberculous mycobacterial (NTM) disease, fungal pneumonia, *Pneumocystis* pneumonia, viral pneumonia, and parasitic infections. Use of prophylaxis intercalarily to antiretroviral therapy (ART)

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and highly active antiretroviral therapy (HAART) for medication are also explained in this study.

PATHOGENESIS

The entire respiratory tract is exposed to a myriad of innocuous and pathogenic particles, including nonpathogenic bacteria, bacterial endotoxins, fungi, and viruses. Systemic diseases such as HIV infections may affect the lung. The significance of smoking should be underlined for HIV-infected patients because it causes increased mortality and decreased quality of life (6, 7).

Risk factors for community-acquired pneumonia are identified as follows: extremes of age (very young or old), male gender, some

populations, daily life issues (extreme alcohol drinking and smoking), medications (e.g., inhaled corticosteroids), supplementary risk factors related to pneumococcal diseases (e.g., myeloma), and underlying comorbid situations (e.g., chronic cardiorespiratory diseases, chronic renal diseases, hepatic situations, diabetes mellitus, malignancy, HIV) (8).

AGENTS

Pneumonia can be classified as bacterial pneumonia, TB, NTM disease, fungal pneumonia, PCP, viral pneumonia, and parasitic infections depending on the agents (Table 1). According to a research conducted, PCP, *Mycobacterium tuberculosis*, *Streptococcus pneumo-*

Table 1. Etiology of pulmonary infections in HIV-infected patients

Etiology		Cumulative incidence
Infectious etiology		97% of pulmonary infiltrates with diagnosis
Bacterial pneumonia		60% of pulmonary infiltrates of infectious etiology
	Streptococcus pneumoniae	70% of bacterial pneumonia
	Haemophilus influenza	10% of bacterial pneumonia
	Staphylococcus aureus	9% of bacterial pneumonia
	Legionella pneumophila	6% of bacterial pneumonia
	Gram-negative bacillus	5% of bacterial pneumonia
PCP		20% of pulmonary infiltrates of infectious etiology
Mycobacteriosis		18% of pulmonary infiltrates of infectious etiology
	Mycobacterium tuberculosis	80% of mycobacteriosis
	Mycobacterium kansasii, MAC,	
	Mycobacterium fortuitum and	
	Mycobacterium xenopi	20% of mycobacteriosis
Virus		5% of pulmonary infiltrates of infectious etiology
	Cytomegalovirus	
	Influenza virus	
	Parainfluenza virus	
	Respiratory syncytial virus	
Fungus		2% of pulmonary infiltrates of infectious etiology
	Cryptococcus	
	Aspergillus fumigatus	
	Endemic fungal infections	
Parasite		0.5% of pulmonary infiltrates of infectious etiology
	Toxoplasma gondii	
	Strongyloides stercoralis	
Multiple organisms		7% of pulmonary infiltrates of infectious etiology
Other		3% of pulmonary infiltrates of infectious etiology
Noninfectious etiology		
	Pulmonary edema	
	Lung cancer	
	Other	

HIV: Human immunodeficiency virus; MAC: Mycobacterium avium complex; PCP: Pneumocystis pneumonia

niae, and *M. pneumoniae* were the most common etiologic agents among HIV-positive patients (9).

Bacterial Pneumonia
Prior studies have shown that bacterial pneumonia is the most common disease in HIV-infected patients, in whom the frequency of bacterial pneumonia is increased by >10 times.

Intravenous drug use, smoking habit, elder age, measurable virus load, and earlier iterative pneumonia are the major risk factors for the progression of bacterial pneumonia in HIV-infected patients. Bacterial pneumonia may develop at any moment in HIV-infected patients, but the CD4 levels stimulate this probability. The median CD4 value that causes the development of bacterial pneumonia is 200/mm³. The median CD4 value causing TB or PCP pneumonia is greater than that causing bacterial pneumonia. On the other hand, the HIV load for TB or PCP pneumonia is lower than that for bacterial pneumonia (10).

Pseudomonas aeruginosa is the frequent cause of community-acquired and nosocomial bacterial pneumonia in hospitalized cases among HIV-infected patients having nominal CD4+ levels (11).

Bacterial pneumonia is more common among HIV-infected patients than among seronegative healthy people. The probability is the highest among CD4 lymphocyte levels under 200/mm³ and among injection drug addicts (12).

Tuberculosis
While *M. tuberculosis* and HIV are independently responsible for substantial human suffering and death, their combined effect has been disruptive in areas where these pathogens dually infect the population (13).

The pathogen *M. tuberculosis* complex has developed various mechanisms to enable its successful coexistence with the human host during the long period of coevolution (14).

HIV-infected patients have significantly higher risk of progressing from latent TB infection to active TB than people without HIV infection.

According to the clinical research by Getahun, the risk for progression to TB disease after *M. tuberculosis* infection is increased among HIV-infected patients (15).

Jones et al. (16) evaluated the clinical and laboratory findings of 97 HIV-infected patients with TB in terms of the number of CD4 cells between the clinical presentation of TB and HIV-infected patients. Extrapulmonary TB was found in 30 (70%) of the 43 patients with lower than or equal to 100 CD4 cells/µL, 10 (50%) of 20 patients with 101 to 200 CD4 cells/µL, 7 (44%) of 16 patients with 201 to 300 CD4 cells/µL, and 5 (28%) of 18 patients with greater than 300 CD4 cells/µL (p=0.02).

Nontuberculous Mycobacterial Disease
Nontuberculous mycobacteria commonly cause disseminated disease in HIV-infected patients. *M. avium* complex (MAC) and *M. kansasii* are major causative agents for the disseminated NTM situations. Disseminated MAC infection necessitates separation of the agent from the sterile tissues for definite diagnose (1).

Similarly, in the research of Jones et al. (16), mycobacteremia were found in 18 (49%) of 37 patients with lower than or equal to 100 CD4 cells/µL, 3 (20%) of 15 patients with 101 to 200 CD4 cells/µL, 1 (7%) of 15 patients with 201 to 300 CD4 cells/µL, and none of 8 patients with greater than 300 CD4 cells/µL (p=0.002).

Fungal Pneumonias
Pneumocystis, *Cryptococcus*, *Histoplasma*, and *Coccidioides* are the most common agents, but *Blastomyces*, *Aspergillus*, and *Penicillium* could be counted as well. Cryptococcosis occurs at last stages of HIV disease. *C. neoformans* is an epidemic agent in HIV-infected patients in the United States of America (USA) and frequently manifests as meningitis. Pneumonia is less common. Serum cryptococcal antigen tests can be functional for diagnosis, and the agent is readily cultured from sterile tissue (1).

The most frequent cause of mycosis is histoplasmosis in HIV-infected patients. In the development of widespread histoplasmosis and coccidioidomycosis, CD4 values are usually below 100/mm³, while the value is above 250/mm³ in case of lobar pneumonia. Blastomycosis is not very common in HIV-infected patients; however, it can cause severe complications (10).

Pneumocystis Pneumonia
P. jirovecii is the most common microorganism causing pneumonia. It also causes PCP. PCP can be diagnosed with the microorganism by sputum, bronchoalveolar lavage (BAL) fluid, or tissue biopsy. The first things that should come to mind with cavitations in the upper lobe are PCP and TB. *P. jirovecii* can colonize airways with no symptoms.

PCP primarily occurs in HIV-infected patients whom the CD4 value is below 200/µL. The serum microorganism load of it is generally greater than 10 000 copies/µL (10).

Viral Pneumonia
The main causative agent of viral pneumonia is the herpes virus family that is resistant to treatment (2). Influenza is the most frequent infectious agent of pulmonary diseases in HIV-infected patients (10).

Cytomegalovirus (CMV) primarily occurs in HIV-infected patients when their CD4 value is below 50/mm³. A unique difficulty is caused by the entity of CMV with another microorganism located in BAL, mostly *P. jirovecii*. The function of this entity has not yet been explained. This complex situation has not been assumed as criteria for the diagnosis of CMV pneumonia (10).

Parasitic Infections
Parasitic infections can cause significant morbidity and mortality in HIV-infected people. Causative microorganisms are *Toxoplasma gondii*, *Strongyloides stercoralis*, *Cryptosporidium*, and *Microsporidium*. *T. gondii* is the most frequent cause of pneumonia. The secondary frequent reason is encephalitis. Pulmonary toxoplasmosis causes pneumonia in the people with CD4 value lower than 100/mm³. The identification of respiratory toxoplasmosis is commonly verified via bronchoscopy with BAL. A small number of case reports of pneumonia are owing to *S. stercoralis*, *Cryptosporidium*, and *Microsporidium*, which arises in disseminated form (10).

TREATMENT AND CLINICAL CAUSE
According to clinical research, the treatment of pneumonia consists of prophylaxis, which changes the agents of pneumonia with ART or HAART (17). An obvious decrease has been shown in the incidence

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of bacterial pneumonia as a result of using combination ART that contains protease inhibitors (PIs). HAART, in particular, prevents respiratory infections such as the kind of pneumonia stated above by protecting immunity.

HIV-infected patients can expect improved morbidity and mortality with early initiation of ART, even in the presence of acute OI. This benefit can be detected as early as 6 months after the initiation of therapy (17). The progress of using ART is shown in Figure 1.

Since the development of HAART and the use of prophylaxis drugs, hospitalizations of HIV-infected patients because of OIs were decreased to a greater extent (18, 19).

Bacterial Pneumonia

Proinflammatory cytokine/chemokine of BAL appears to be a valuable diagnostic tool to avoid unnecessary invasive diagnostic procedures or treatments (20). The clinical response to antibiotics and its outcomes are similar in HIV-infected patients and HIV-noninfected persons with bacterial pneumonia (21).

The most frequent causative agent of community-acquired pneumonia is *S. pneumoniae* in these patients. In contradiction to a clinical study, results show that vaccination with the pneumococcal polysaccharide vaccine has increased the risk of pneumonia. Despite this, many studies have stated advantages from vaccination with the 23-valent pneumococcal polysaccharide vaccine in these patients.

Studies have demonstrated a decrease in pneumococcal bacteremia by vaccination. A large number of HIV experts consider that the possible profit of pneumococcal vaccination offsets the hazard. The Centers for Disease Control (CDC) and the Infectious Disease Society of America (IDSA) guidelines advise that a unique dose of polysaccharide vaccine should be vaccinated at the first opportunity following the diagnosis of HIV-infected patients. The second vaccination can be contemplated for patients who were primarily vaccinated when their CD4 values were lower than 200/µL and whose CD4 values have raised above 200/µL in reaction to ART (10).
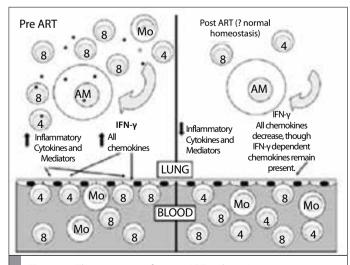

Figure 1. The progress of using ART in lung immune response. Model of HIV and lung immune response before and after antiretroviral treatment
AM: Alveolar macrophage; ART: antiretroviral therapy; HIV: human immunodeficiency virus; MO: monocyte; 4: CD4 T cell; 8: CD8 T cell

Similarly, Heffernan et al. (22) demonstrated that there was a 57% reduction in invasive pneumococcal disease frequency among HIV-infected patients throughout the first 5 years after the beginning of ART.

Feikin et al. (23) have shown that bacterial pneumonia rates were up to 25-times higher among HIV-infected adults than among healthy persons; this rate increases as the CD4+ T-cell amount decreases. Cohort studies have shown that in developed countries, HAART has the most consistent effect on reducing pneumonia. In prospective cohort and case–control studies from these regions, pneumococcal polysaccharide vaccine reduced pneumococcal disease in certain subgroups, particularly those with higher CD4+ T cells/µL. In patients with lesser than 200 CD4 T cells/µL, antimicrobial prophylaxis was usually effective in reducing pneumonia. In sub-Saharan Africa, randomly controlled trials concluded that co-trimoxazole prophylaxis reduced the frequency of bacterial pneumonia, but pneumococcal polysaccharide vaccine avoided neither pneumonia nor invasive pneumococcal disease. Although it is not totally evaluated yet, based on experience in industrialized nations, the use of HAART in Africa may have a significant effect in preventing bacterial pneumonia.

Sullivan et al. (24) have shown that ART was significantly associated with the reduction in the incidence of bacterial pneumonia in HIV-infected patients.

PI treatments (for example, ritonavir) can result in systemic accumulation of inhaled corticosteroids and may increase the risk of pneumonia by exacerbating the toxicity of this treatment in HIV-infected inhabitants with asthma or chronic obstructive pulmonary disease (25).

Pneumocystis Pneumonia

In half of the HIV-infected patients, PCP can even arise in early stages of disease. In this condition, the meaning of initial identification becomes important in potential individuals (10).

The precursor of accomplished therapy of PCP is increased levels of S-adenosylmethionine because low levels of S-adenosylmethionine are a sign of PCP.

Trimethoprim–sulfamethoxazole (TMP–SMX) is the counseled initial regimen for both therapy and prophylaxis PCP, and a review suggests that clindamycin added to primaquine is a successful secondary option. Apprehension has been increased over the possible progression of TMP–SMX drug resistance in the form of nonsynonymous mutations in the human *Pneumocystis* dihydropteroate synthase (DHPS) gene. These mutations have only been detected in human *Pneumocystis* (1).

Prophylaxis needs to be administered only during periods of high risk. Molecular studies have defined some mutations in the goal of sulfa drugs that seem to symbolize the development of resistance in *P. carinii*. Resistance to atovaquone, a second-line agent, may also be developed (26).

Therapy can be initiated prior to diagnosis exactly for the reason that agents endure in patients samples for a long time following therapy. TMP–SMX is the best regime during therapy. The advised period of treatment for PCP is 3 weeks. After the end of treatment, cases should be instantly medicated on prophylaxis.

Definite damages of *P. jirovecii* include mutations in the DHPS gene. It is related to the period of use of TMP–SMX prophylaxis. In contrast,

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it is not associated with treatment success. Lately, a clinical trial recommended that the intermission of prophylaxis may be harmless in HIV-infected patients with CD4 values of 101–200 per mL and depressed HIV load (10).

In a different study in which the study population had CD4 levels of 200 to 499 mm³, the frequency of PCP raised by 40% on average per year. For the study population with CD4 levels <200 mm³, rates of bacterial pneumonia and PCP were elevated and the rate of other pulmonary OIs increased with time (27).

In another study, the clinical features of 10,549 episodes of PCP in patients with AIDS were compared with those of 46 episodes in patients with other immunosuppressive diseases. The survival rate was similar in the groups (12.4% vs. 15.2%, p=0.64). PCP exists as a further sneaky disease course in patients with AIDS, and treatment is complicated by frequent adverse reactions (28).

In addition, the incidence of OI in HIV-infected patients has markedly decreased since the advent of HAART. Regardless of this, there are numerous PCP patients at the reference center even now. In total, 104 patients, who are pathologically proven cases, were included in the study. Of these, 69% were active material abusers and 50% had prior information of HIV infection. Less than 5% of patients were on HAART or PCP prophylaxis at study admission. The whole mortality rate was 14%. Among dischargedpatients, 65% received HAART treatment and 59% of these attained a viral load of less than 1000 copies/mL in the year when they were dismissed. Thus, patients are still being admitted with PCP in the HAART era. An aggressive approach toward HIV identification and substance abuse treatment may decrease admissions to the hospital for PCP and may improve response to HAART therapy (29).

Tuberculosis and Nontuberculous Mycobacterial Disease
In HIV-infected patients, TB treatment is similar with to that in noninfected patients.

The results of a review are in accordance with the systematic review on the effectiveness of rifampicin added to pyrazinamide for the avoidance of TB infection that included both HIV-infected and non-infected people (30).

Tuberculosis therapy can be complicated because of drug co-actions and lapping toxicities, while treatment for HIV and TB is concurrently managed together. Rifamycins stimulate hepatic CYP3A4 enzymes that can expedite the metabolism of PIs and non-nucleoside reverse transcriptase inhibitors (NNRTIs) principal to the noncurative stage of these antiretroviral medicines. Rifampin should not be used in individuals on PIs principle treatments. Although rifampin stages of all the NNRTIs, previously it has fewer influenced. Rifabutin is a choice instead of rifampin that can be managed with PIs or NNRTIs. Latest trials recommend that rifampin has a significant function in the therapy of HIV-related TB. If it is not used in the continuation phase, the recurrence rate is increased by 2–4 times. Rifampin-established therapy and efavirenz-established ART are possibly the favored therapy methods for HIV-related TB. Many studies have shown the clinical advantage of starting ART though TB treatment in the early periods. Initial HAART diminished HIV infection improvement and mortality in HIV-infected HAART-inexperienced patients with TB and with a CD4 value of <50 per mm³. HAART can be used in patients with a CD4 value of >50 per mm³ after 2 months following the start of TB therapy. HAART should not be postponed until the end of TB treatment for pa-

tients with CD4 values ≤ 500 per mm³. The possibility of immune reconstitution inflammatory syndrome (IRIS) was increased in patients who received ART in the initial phase of treatment. A previous study demonstrated that therapy by prednisone for 4 weeks decreased the occurrence of IRIS in patients with TB who received HAART as TB medication. Therapy alternatives for latent TB comprise isoniazid daily or twice a week for 9 months. The latest study demonstrates that a 3-month process of isoniazid in combination with rifamycin may be a useful option.

Respiratory diseases caused by *Mycobacterium* sp. are observed with an increased incidence in HIV-infected patients as well.

Treatment occurs with ethambutol and a macrolide, generally clarithromycin. Rifabutin, ciprofloxacin, or amikacin can be added in case of disseminated infection. It is possible to stop treatment in patients with maintained inhibition of HIV duplication and CD4 values >100/mL for >6 months (10).

In Nahid et al. (31) study, HIV-infected patients who received a 6-month rifamycin-based course of TB treatment or who received intermittent therapy had a higher relapse rate than HIV-infected patients who received longer therapy or who received daily therapy, respectively. Standard 6-month therapy may be insufficient to prevent relapse in HIV-infected patients.

Retrospectively studied HIV-infected patients with pulmonary isolated *M. kansasii* had a lower CD4 count [hazard ratio (HR), 1.6; 95% confidence interval (CI), 1.1–2.3] and positive smear microscopy (HR, 2.8; 95% CI, 1.3–6.1) that were associated with mortality, whereas ART (HR, 0.3; 95% CI, 0.1–0.8) and *M. kansasii* treatment (HR, 0.4; 95% CI, 0.2–0.9) were associated with survival. ATS criteria did not predict mortality (HR, 0.9; 95% CI, 0.4–1.9). This study suggested that withholding treatment in HIV-infected patients with respiratory isolated *M. kansasii* should only be considered with negative smear microscopy, few positive cultures, and mild immunosuppression (32).

Furthermore, there is growing evidence of the benefits of early initiation of ART in subjects co-infected with TB. In a recent study by Abdool Karim et al. (33), there was a 56% reduction in mortality when ART was initiated during TB therapy compared with that when it was delayed until the completion of TB therapy.

Despite the availability of ART, many HIV-infected patients are still dying from TB, and measures to control OIs such as TB are therefore particularly important (34).

Viral Pneumonia
Ganciclovir has been the main anti-CMV drug using intravenously despite the issues associated with its use, such as effectiveness, toxicity, low oral bioavailability, and drug resistance. Further trials have shown that the use of foscarnet and cidofovir should be restricted because of their nephrotoxicity. Combined treatment with ganciclovir and foscarnet for drug-resistant agents has been used. Some new medicines have potential, such as the methylenecyclopropane nucleoside analogs and benzimidazole. There is a need for future studies for the treatment and prophylaxis of immunoglobulins (35).

Patients are protected from influenza pneumonia via vaccination. Lately presented clinical trials have demonstrated that HIV-infected patients with A/H1N1 disease who are kept under control on HAART had comparable medical result to controls. Influenza vaccination for

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HIV-infected patients is suggested by the CDC and IDSA once a year. Despite this, proposal has not recognized general care. A previous clinical study demonstrated a 20% total decrease in the probability of respiratory symptoms and 100% defense against influenza in the vaccinated group in comparison with the placebo group. Some reviews accomplished that vaccination of these patients may be resourceful despite unpredictable antibody reactions. The effects of human metapneumovirus infection in these patients have not been identified yet (10).

Fungal Pneumonia

Prophylaxis in fungal infections treatment is based on amphotericin B and triazoles. The conditions that are required to be met before suppressive azole therapy is discontinued are as follows: itraconazole therapy for 1 year, negative blood cultures, histoplasma serum antigen of 2 enzyme immunoassay units, CD4 cell count >150 cells/mm^3, and HAART for 6 months (36).

It would be possible to discontinue secondary prophylaxis after 12 months of therapy in patients with *focal coccidioidal pneumonia* who had responded to antifungal therapy were receiving ART and had a CD4 count cell of >250 cells/mm^3. However, patients who have diffuse pulmonary disease or disseminated coccidioidomycosis should continue therapy indefinitely. Patients receiving ART who have had a CD4 cell count of >150 cells/mm^3 for ≥6 months can discontinue itraconazole therapy for blastomycosis after a minimum of 1 year (36).

The recommended treatment is amphotericin B followed by oral itraconazole in patients infected with *Penicillium marneffei*. These patients should be administered secondary prophylaxis with oral itraconazole. Discontinuing secondary prophylaxis for penicilliosis is recommended for patients who receive ART and have a CD4 count of >100 cells/mm^3 for ≥6 months (36).

While *Cryptococcosis* arises in HIV-infected patients, the proposed therapy is amphotericin B in combination with flucytosine for 14 days, followed by fluconazol. Secondary prevention can be stopped while there is a maintained escalation of CD4 values to ≥200/mm^3 after HAART. After the development of HAART, the incidence of cryptococcal infection and other endemic fungal infections is suggested to decrease (10).

CONCLUSION

Pneumonia is the leading cause of morbidity and mortality in HIV-infected patients universally. In this paper, we identified pathogenesis, causative agents, treatments, clinical course, and treatment of pneumonia in HIV-infected patients. Improved ART and prophylaxis for OI developments have been used in combination to get a better life quality in HIV-infected patients and their survival. Despite new developments, what awaits patients and doctors in the coming days remains unknown (37). Few articles have described the significance of HAART on bacterial pneumonia. However, many clinical trials have shown that HAART would be related to a reduction in the occurrence of bacterial pneumonia (10). Data from a randomized trial of continuous versus intermittent ART showed that the risk of pneumonia was significantly higher among patients who received intermittent treatment. In this study, it is explained that the ART reduces the risk of bacterial pneumonia, even for people with CD4 cell counts of ≤500 (38). Despite the lack of consensus about the use of vaccines for protective treatment of pulmonary disease in patients with HIV, several studies have shown that vaccines are also helpful in specific conditions.

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